

Case Report —

POSSIBLE OUTBREAKS OF
FUSARIOTOXICOSIS IN AVIANS

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SUMMARY

A disease syndrome characterized by raised yellowish-white lesions in the oral cavity was observed in several commercial broiler flocks. Growth rates were depressed, and 10% died. Fowl pox was ruled out by infectivity tests in susceptible birds with lesion material. A similar outbreak occurred in fancy pigeons fed visibly moldy feed. The oral lesions in pigeons contained large numbers of avirulent *Staphylococcus epidermidis* and *Escherichia coli*. Identical oral lesions were produced in the laboratory by feeding chickens small concentrations of fusariotoxin T-2 produced by the fungus *Fusarium tricinctum*.

INTRODUCTION

In the past few years we have encountered a disease of unknown etiology in broiler chickens and fancy pigeons characterized mainly by raised yellowish-white lesions of the mouth parts. The lesions would become so severe that the birds could not close their mouths completely and could eat only with difficulty. Inspection of the literature revealed a report that feed inoculated with *Fusarium* strains produced a yellowish-white fibrinous deposit in the mouths of goslings (14). Recently, it has become possible to produce good yield of pure fusariotoxin T-2 from *Fusarium tricinctum* (4), and thus to do controlled experiments with a single chemical entity. This mycotoxin is produced by at least four species of *Fusarium* (5) and by *Trichoderma lignorum* (2), all fungi frequently isolated from moldy cereals.

The chemical structure of fusariotoxin T-2 is 4,15-diacetoxy-8-(3-methylbutyryloxy)-12,13-epoxy- Δ^9 -trichothecen-3-ol (1). Like

most 12,13-epoxy- Δ^9 -trichothecen compounds, it is a potent skin irritant and inflammatory agent (12). In trout, dietary fusariotoxin T-2 causes a rapid sloughing of the intestinal mucosa (11). In rats, feeding T-2 toxin results in a severe inflammation around the nose and mouth, while topical application causes a necrosis of the dermal tissue (11). In broiler chickens, small concentrations of dietary T-2 toxin produce dose-related oral lesions (9).

The present paper describes field outbreaks characterized by oral lesions and points out similarities to the toxicosis caused by fusariotoxin T-2. Increased awareness of fusariotoxicosis by the poultry industry will assist a valid assessment of its importance.

The first outbreak occurred in chickens in an integrated broiler operation. The growers noted the outbreak when the chickens were about 6 weeks old, and we examined the flocks when they were 7 weeks old. The most visible effects of the disease were lesions on the feet and shanks (Fig. 1) and on the head around the eyes (Fig. 2). The lesions, which were covered by granular scabs, resulted from vesicle formation. Not all sick birds had involvement of both legs and eyes. Upon close inspection, however, all affected birds (over 300 were examined), regardless of the severity of the external lesions, had raised yellowish-white lesions or eruptions of

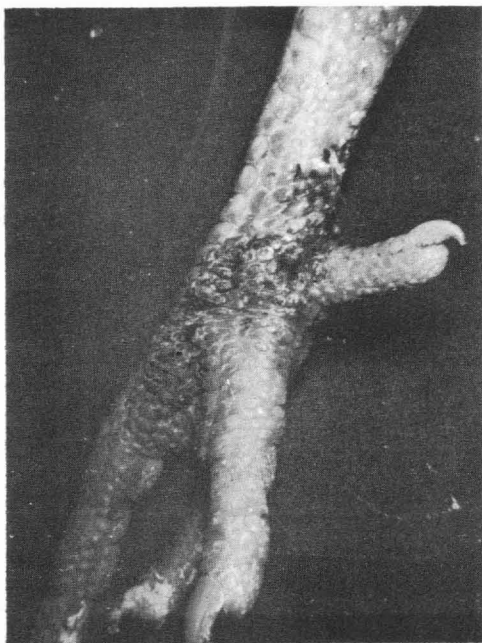


Fig. 1. Foot and shank of a broiler chicken displaying vesicles and scab-covered lesions.

the oral cavity (Fig. 3). The oral lesions consisted of caseous, necrotic material that resembled a pseudomembrane. This material could be teased with forceps from the mouth parts, leaving slightly bloody erosions. Morbidity varied from 10 to 25%. On Necropsy the internal organs appeared normal. Severely affected birds died in a few days, from septicemia apparently developed from secondary bacterial invaders of the lesions. Deaths never exceeded 10% of the house. The survivors were normal in appearance and growth rate.

Since these symptoms resemble rather closely those seen in fowl pox (7), an infectivity test (3) was performed. Twenty birds challenged with the lesion material from twenty affected birds developed no signs of fowl pox or other disease. Susceptibility was proved by the development of lesions after injection of authentic fowl pox virus. This suggested that the disease was not infectious. This conclusion is supported by the spotty occurrence of the disease. Not all flocks had the disease, and even adjacent houses were seldom affected. Such erratic occurrence is characteristic of mycotoxicoses.

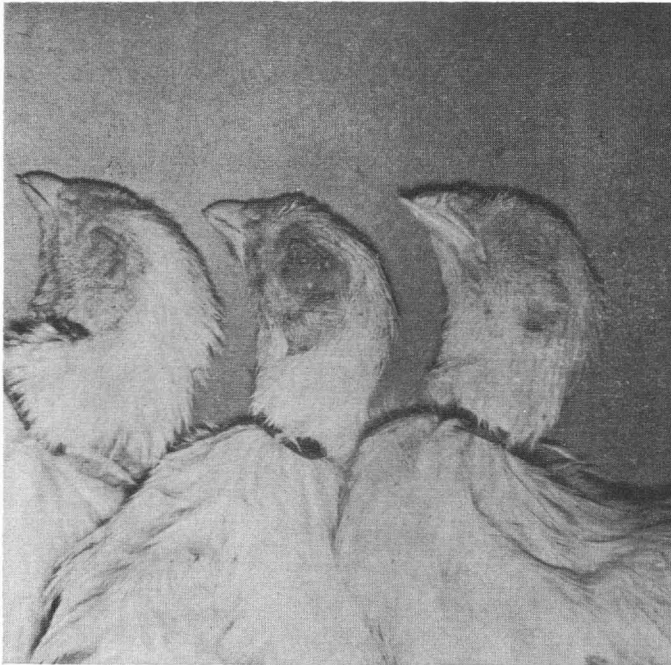


Fig. 2. Heads of broiler chickens displaying lesions.

Another disease which this outbreak resembled somewhat is vesicular dermatitis (sod disease). The lesions of sod disease, which received its name because of its prevalence in chickens ranging over unbroken prairie sod (13), are apparently limited to the feet and shanks. However, several mycotoxicoses have been caused by moldy grass and hay (8). Also, Perek (15) reported outbreaks of vesicular dermatitis, including lesions on the head, in Israel, and reproduced the lesions in chickens by giving feed contaminated with the fungus *Cladosporium herbarum*.

The outbreak in broilers which was referred to us occurred in 1966, while necessary analytical techniques for mycotoxins were still largely undeveloped; hence no direct investigation of the etiology was possible. Because of the presumption that the outbreak was caused by highly irritating mycotoxins produced in the feed and/or litter, the litter of the affected houses was treated with quick-lime, and a shallow layer of new shavings was added on top. The management program was improved to reduce the likelihood of moldy feed, and there have been no further outbreaks in that operation.

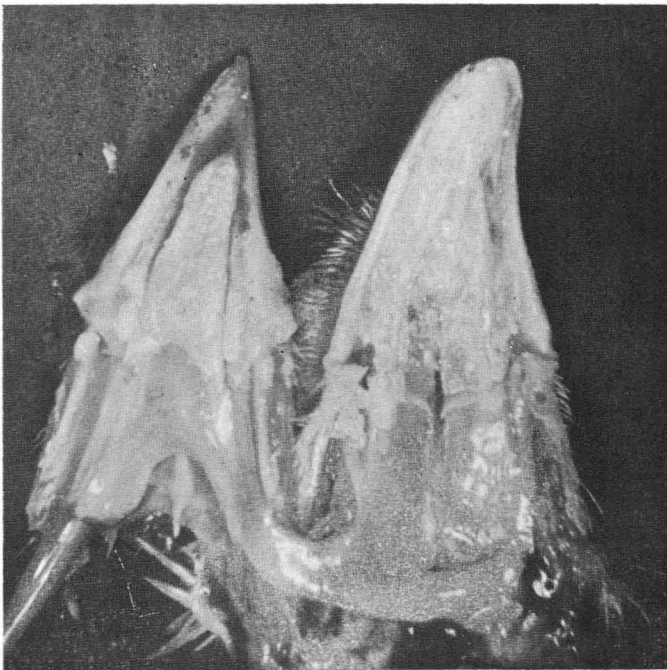


Fig. 3. Mouth parts of a broiler chicken displaying characteristic raised yellowish-white lesions.

Another outbreak occurred during 1970 in a flock of pigeons kept by a pigeon fancier. This fancier was unaware that moldy feed presented a health hazard to avian species, and he fed his pigeons visibly moldy feed for approximately two months. The pigeons developed severe mouth lesions identical to those seen earlier in broilers. *Escherichia coli* and *Staphylococcus epidermidis* were isolated readily from these lesions, but they proved nonpathogenic when inoculated into day-old chicks. *Candida albicans*, which can produce oral lesions (6), could not be isolated. Microscopic examination did not reveal any protozoa such as *Trichomonas gallinae*, which causes trichomoniasis (canker) in pigeons (10). On necropsy, the internal organs appeared normal, including the

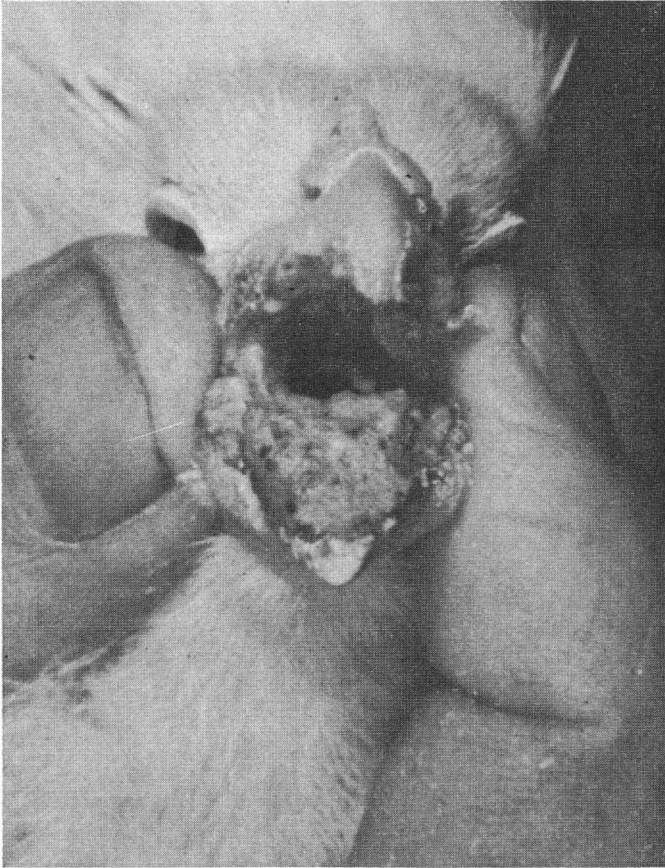


Fig. 4. Oral lesions produced in young broiler chicken in three weeks by feed contaminated with fusariotoxin T-2 at 16 ppm.

esophagus, crop, and liver, which are involved frequently in trichomoniasis (10). Also, the oral lesions were not as large as those usually seen in pigeon canker. About one-third of the flock of over forty pigeons was affected and about 5% died. There were no lesions on the shanks, feet, or heads of the pigeons, which were housed on wire. Unfortunately, the feed had been consumed about a week before the case came to our attention, so it could not be analyzed for T-2 toxin or *Fusarium*. The survivors became asymptomatic about four weeks after being placed on fresh feed.

Fig. 4 shows an example of the oral lesions produced in three weeks by feed containing fusariotoxin T-2 at 16 ppm. All the mouth parts of this bird were affected, and they were so swollen that the bird could not close its beak. These raised yellowish-white lesions could be teased from the underlying tissues with only slight bleeding. Fig. 5 shows the inside of the upper beak of birds receiving 4 ppm. The lesions were manifested mainly as V-shaped streaks. At 1 ppm the lesions were restricted to isolated spots on the margin of the tongue and the roof of the mouth. The growth rate was normal in birds receiving 1 ppm, but was depressed at 4 and 16 ppm. These data were obtained with forty birds per dose level.

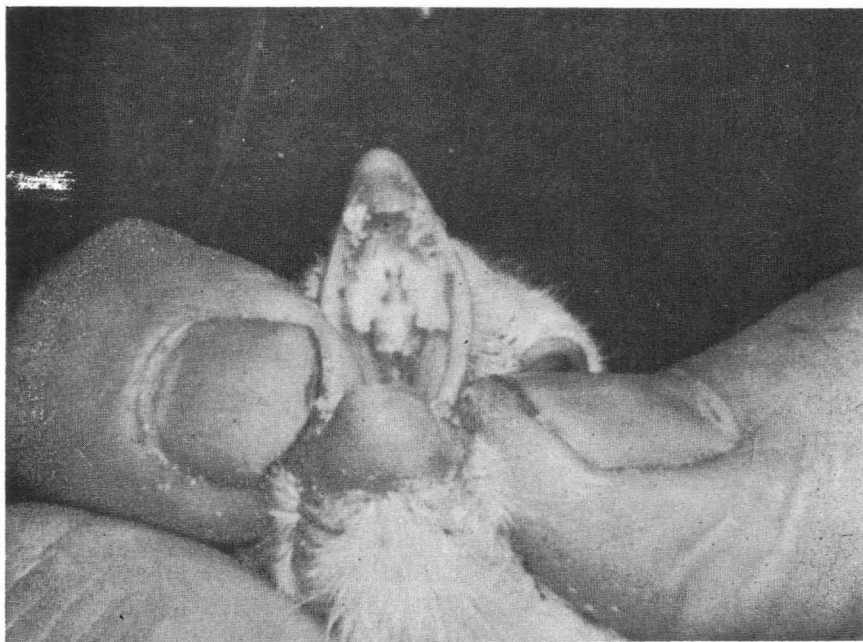


Fig. 5. Lesions on the hard palate produced in three weeks by feed containing fusariotoxin T-2 at 4 ppm.

Microscopic examination of stained sections of the mouth parts of affected birds revealed a severe infiltration of the underlying tissues by granular leukocytes. The surface of the lesions was composed of fibrinous material which was being sloughed. The intermediate layers had pockets containing cocci and rods. *Escherichia coli* and *Staphylococcus epidermidis* were isolated in large numbers from the lesions. Administering the isolated bacteria to control birds in groups of forty with or without scarification of the mouth parts did not produce lesions or impair growth rate. *Candida albicans* could not be isolated from either the mouth or the crop (Wyatt, Weeks, Hamilton, and Burmeister, unpublished results). The internal organs were normal grossly and there were no lesions on the feet, shanks, or heads. The oral lesions produced in the laboratory by incorporating pure fusariotoxin T-2 into feed appeared identical to those seen in field outbreaks in chickens and pigeons.

Thus, it appears that T-2 toxicosis is characterized by easily recognized oral lesions which can become so severe as to interfere with normal eating habits. The lesions seen on the feet, shanks, and head of the broilers in the first outbreak do not appear to be the result of dietary fusariotoxin T-2 but are possibly the result of toxin in the litter. An equally likely possibility is that these external lesions are the result of another mycotoxin, such as that produced by *Cladosporium herbarum*, which causes vesicular dermatitis (15). It seems safe to say that highly irritating mycotoxins can cause topical eruptions severe enough to endanger the health of poultry. The oral lesions appear characteristic enough to be useful in diagnosis, and they occur at small concentrations of the mycotoxins. Awareness of these toxin-induced oral lesions by workers in the poultry industry should help in assessing the severity of the mycotoxicosis caused by fusariotoxin T-2.

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